
 COMMENTS AND
 RESPONSES

**Response to
 Comment on:
 Kim et al.
 Prospective Study
 of Serum
 Adiponectin and
 Incident Metabolic
 Syndrome:
 The ARIRANG Study.
 Diabetes Care 2013;
 36:1547–1553**

We thank Dr. Kawada (1) for his comments in response to the ARIRANG study findings on the predictive ability of adiponectin for metabolic syndrome (2). Dr. Kawada raised concerns about the appropriateness of logistic regression because there was some variability in the follow-up times of study participants even though the follow-up period was relatively short (median 2.4 years, interquartile range 2.0–2.9 years). In response, we repeated the analyses using a Cox model with very similar results. The hazard ratio (95% CI) for metabolic syndrome comparing adiponectin quartiles 2–4 to the first quartile in model 3 were 0.57 (0.37–0.88), 0.71 (0.47–1.08), and 0.31 (0.18–0.54),

respectively, in men, and 0.64 (0.43–0.94), 0.64 (0.44–0.94), and 0.60 (0.40–0.92), respectively, in women.

Second, Dr. Kawada questioned the approach to confounder adjustment. We used three models with progressive degrees of adjustment, including different combinations of the confounders referred to by Dr. Kawada, but the point estimates changed little with progressive adjustment indicating that the results were reasonably robust to the inclusion of available confounders. Dr. Kawada also pointed out the limitations of homeostasis model assessment of insulin resistance as a marker for insulin resistance in subjects with fasting plasma glucose ≥ 140 mg/dL. In the ARIRANG study, only 25 subjects had fasting plasma glucose ≥ 140 mg/dL at baseline (1.2%) and exclusion of these participants did not change the results.

Finally, Dr. Kawada was concerned about the potential bias induced by losses to follow-up. In the ARIRANG study, 74.6% of study participants attended the follow-up visit, and participants who attended the follow-up visit had similar values of baseline demographic and laboratory findings compared with those who did not attend the follow-up visit and none of the differences was statistically significant. Although we cannot exclude selection bias, the comparability of both groups makes it less likely that selection bias may substantially affect the conclusions.

In summary, our study provided robust prospective evidence that serum adiponectin was an independent protective factor for the development of metabolic syndrome.

Additional studies should further corroborate our findings on the usefulness of serum adiponectin in cardiometabolic risk stratification tools.

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References

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